tightly binds the GpIIb-IIIa receptor, so the pool of circulating, unbound drug is negligible. Transfused platelets therefore remain functional. The timing of platelet administration is as yet unclear; we transfuse after weaning from cardiopulmonary bypass. Additionally, because abciximab increases the activated clotting time in patients receiving heparin, we recommend a reduced heparin dosage for patients undergoing cardiopulmonary bypass, with the caveat that optimal heparin dosage in the presence of GpIIb-IIIa inhibitors is as yet undefined. We currently use a 150-μg/kg heparin dose and target an activated clotting time of 500 seconds. It is also critical that surgeons recognize the importance of delay between administration of the particular GpIIb-IIIa inhibitor and the operation; if possible, waiting for a period equivalent to the effective circulating half-life of the drug should be considered. This factor takes on additional relevance as newer, shorter-acting GpIIb-IIIa reach the market (tirofiban and eptifibitide have recently been approved by the Food and Drug Administration). The use of various "off-pump" techniques to avoid cardiopulmonary bypass and full heparinization should be considered when anatomically feasible and appropriate. Finally, delay of the operation until after clearance of the GpIIb-IIIa inhibitor is a reasonable strategy.

Alvarez[1] recommends the use of aprotinin as an adjunct to hemostasis in patients requiring an emergency operation in the face of GpIIb-IIIa inhibition. Aprotinin reduces mediastinal bleeding in patients at high risk; however, its salutory effect on platelet function is mediated through the GpIb receptor, affecting platelet adhesion rather than aggregation. Although it is tempting to try anything when faced with this difficult clinical problem, further information regarding the interaction between aprotinin and GpIIb-IIIa inhibitors is certainly needed.

The importance of the platelet thrombus in acute coronary ischemic syndromes and the ability to affect this process has been called a "new frontier in myocardial reperfusion therapy."[3] For the significant numbers of patients who face cardiac surgery after profound platelet inhibition, however, these inhibitors of platelet aggregation are a 2-edged sword. In light of the number of percutaneous interventions performed annually in the United States and the number of various GpIIb-IIIa inhibitors (oral and intravenous) coming to market, the magnitude of this problem is certain to rise. To treat patients in these difficult cases, cardiac surgeons will need an effective management strategy that is based on an understanding of the pharmacodynamics of the different platelet inhibitors and the pathophysiology of platelet adhesion and aggregation.

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Heart reduction surgery can reconstitute the residual stress-strain state of the left ventricle

To the Editor:

Left ventricular (LV) reduction is a new surgical therapeutic option that has been devised and advanced by Batista and his associates[1] for the treatment of end-stage dilated cardiomyopathy. Resection of a large portion of the free wall of the LV muscle mass results in significant reduction in the LV cavity size and may improve ejection fraction. The ultimate goal of the procedure is to reduce the diameter of the left ventricle to restore a "more optimal" physiologic volume/mass relationship.[1,2] Regrettably, the scientific foundation for this operation is devoid of laboratory data. Only recently has there been an attempt to formulate a physiologic rationale for this operation[2] using a multiple-compartment elastance model to simulate the global effects of volume reduction on cardiac pump mechanics. The observed clinical benefits (improved ejection fraction, enhanced elastance) of ventricular mass reduction were based on sound theoretic argument.[2] What remains is how to explain the apparent increase in ventricular performance that results from variable removal of the myocardium.

Many of the mechanisms of action attributed to volume reduction are rooted in long-standing belief that emphasizes wall stress as the offending cause and the target variable to be surgically manipulated.[1,3] However, equating normalization in chamber size with reduction in wall stress may be expedient but not necessarily the principal mode by which this procedure restores myocardial function.[1,3]

On closer examination, the argued claim of reduction in active wall stress is unfounded on both theoretic grounds and clinical findings, as demonstrated by a near doubling in end-systolic elastance after the volume reduction procedure. It is hard if not impossible to reconcile the enhanced contractile function with significant attenuation of active wall stress. An equally compelling alternative argument for the observed improvement in contractile function and cardiac energetic efficiency can be formulated. This argument is centered about the concept of residual wall stress-strain state of the myocardium.

A fundamental property of the heart is its ability to engage all regions and transmural layers of myocytes in a collective and nearly uniform pattern of contraction. A common basic property of many solid and hollow organs is persistence of residual strain (and hence stress) when all external loads (cavity pressure) are removed. In elastic structures such as blood vessels and heart chambers, this property can be read-

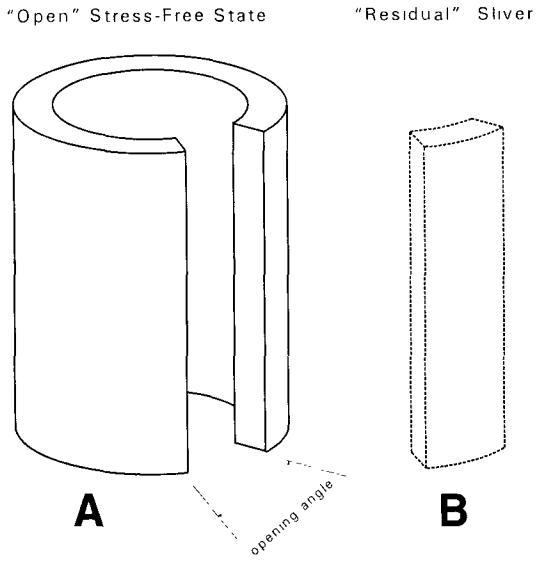

Fig 1. Cylindrical model **(A)** of the left ventricle at zero-stress state. The resulting opening angle, in response to a longitudinal cut, is indicative of the residual pre-strain and associated stored compressive/tensile stresses. Volume reduction surgery in effect mimics the strain-stress state by resecting a sliver **(B)** of muscle. Transformation of the "open" unstressed state to closed pre-stressed configuration is a natural by-product of the procedure.

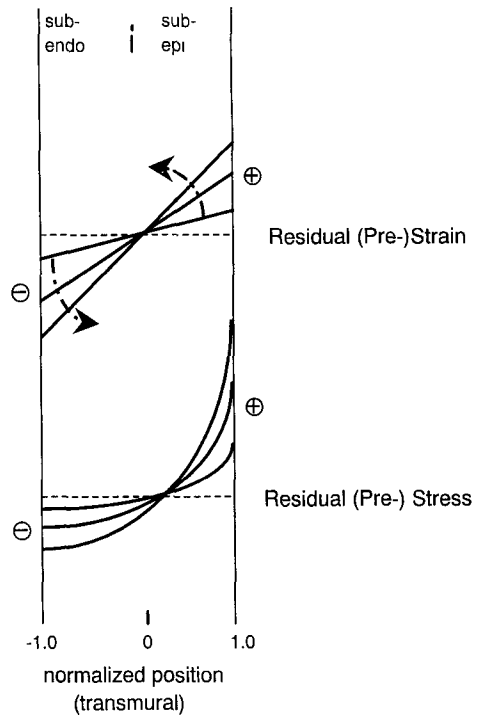

Fig 2. Idealized plots of transmural (endocardium-epicardium) residual strain and stress. *Arrows* describe the increasing degree of deformation (pre-strain angle of opening) at zero-stress predicted for a cylindrical model of the heart (Fig 1). Note that the strain/stress are compressive (-) in the subendocardial layer and tensile in the subepicardial layer. (Adapted from Fung YC. Biomechanics-Circulation. New York: Springer-Verlag; 1980. p 80. Reproduced with permission.)

ily observed when a radial cut is made to equatorial cross-sectional rings, revealing the zero-stress configuration,[5] manifesting an opening in the muscle ring (Fig 1). In general, the residual strain distribution[4,5] in the heart is such that it exhibits a compressive circumferential stress in the inner layer of the ventricle and a principally tensile stress in the subepicardium (Fig 2). As a point of reference, one would expect a greater increase in the residual stress, that is, greater opening angle, to accompany concentric hypertrophy. Conversely, a dilated, volume-overloaded heart would exhibit a decrease in its tendency to "spring open" (Fig 1), leading to a state devoid of residual stress. A potential benefit of an elastically preloaded ventricular configuration is that it gives rise to a gradient in transmural sarcomere length at the onset of diastole that normalizes as the ventricle fills.[5] Naturally arising pre-strain may help redistribute the transmural gradient in LV wall stress such that myocytes located in a different region are "loaded" optimally, resulting in greater efficiency of contraction.[6]

We have come to appreciate that the pressure in the left ventricle during the relaxation and filling phase is determined not only by the viscoelastic properties of the myocardium, but also by the active muscle-borne restoring forces. The phenomenon of "diastolic suction" is coupled, in part, to the ability of the heart to eject beyond the so-called "unloaded" volume (LV pressure ~ 0). Reinstating these favorable conditions, that is, smaller end-systolic volume, whereby an increase in elastic recoil is made operational again can aid in ventricular filling. One would also expect that any form of effective enhancement of contractile performance will have additive benefits in reinstituting the active diastolic filling phase. Clearly, the pre-stressed LV chamber is positioned favorably to "spring back" and, in the process, contribute to the dynamics of LV filling phase. This may explain how end-diastolic compliance can be improved in this patient population despite reduction in chamber size and the resultant, relatively thickened, wall.

The volume reduction procedure has the potential to re-establish the strain-stress state of the LV chamber by surgically mimicking and reconstituting the zero-stress state. By staging and recreating what would be the equivalent of a zero-stress opening arc (Fig 1) seen in healthy hearts, surgical remodeling can recreate, within limits, the residual strain/stress state of the dilated resized heart. A natural by-product of resecting a variable sliver of the LV free wall (Fig 1) is that when the LV chamber is reconstructed the resulting end-effect is reduced diameter, giving rise to a relatively thickened LV wall, necessitating compression of the subendocardial layers and stretching of the subepicardial layers. Importantly, transmural distribution of residual stress is

expected to affect not only the passive and active ventricular properties but also a number of related attributes of cardiac function, such as preload-recruitable stroke work, myocardial energetics, and coronary flow dynamics. The restitution of residual strain-stress may prove to be applicable and contribute to the efficacy of cardiomyoplasty. In particular, recent preliminary data suggest that the benefits (reverse remodeling) observed may be related to passive constants (girdling) of the heart and not to systolic augmentation per se. Clearly the intended benefits of restoring the residual strain/stress are disease-specific and may not be amenable to surgically achieving the ideal in terms of the regional and global structure conformation that may be required. Depending on the prevailing LV geometry and chamber size, the reconstructed heart may not reach the desired degree of pre-strain. This may explain, in part, the variable results achieved clinically thus far [3] It must be recognized that this proposed conceptual framework and theoretical approach remain unproven, requiring basic laboratory supporting data. Moreover, the analysis presented is confined to circumferential strain-stress consideration. A 3-dimensional mapping of the residual (end-systolic) regional strains would provide important additional information that may help "optimize" the planed surgical remodeling (resection size, shape) of the heart, beyond that of performing a "simple" excision.

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The route of choice for the axillocoronary bypass graft

To the Editor:

I read with great interest the recent article by Bonatti and colleagues, "Axillocoronary Bypass for Severely Atherosclerotic Aorta in Coronary Artery Bypass Grafting."[1] Their approach seems to be an excellent solution to prevent cerebral embolization during the construction of proximal anastomoses while performing coronary bypass operations in patients with a severely atherosclerotic ascending aorta. Of concern, however, is the course they chose to tunnel the graft into the thoracic cavity which, in turn, may limit its patency and the application of this procedure.

Several routes have been previously reported to tunnel axillocoronary grafts into the chest cavity: a subcutaneous course,[2] a subfascial plane,[3] a tunnel through the bed of the second costal cartilage,[4] or a tunnel through the intercostal muscles.[5] Bonatti and colleagues[1] chose the last one.

During quiet, deep, and forced respiration, the external intercostal muscles raise the ribs. During forced respiration the internal intercostal muscles lower the ribs. The combined action of the external and internal intercostal muscles draws the ribs together. This can be felt during insertion of chest tubes while performing digital examination of the pleural cavity when patients suddenly cough. It is no surprise that a patient, reported to have undergone an axillocoronary bypass with tunneling of the graft through the intercostal muscles, died suddenly 3 weeks after the operation.[5]

Before using the axillary artery as a source of inflow to revascularize the coronary arteries, I realized that there was no natural passage to reenter the chest cavity.[6] Therefore I decided to remove the anterior portion of the first rib, which transformed the proximal portion of the axillary artery into an intrathoracic structure (Fig 1). This surgical step requires expertise usually acquired while treating patients with tho-

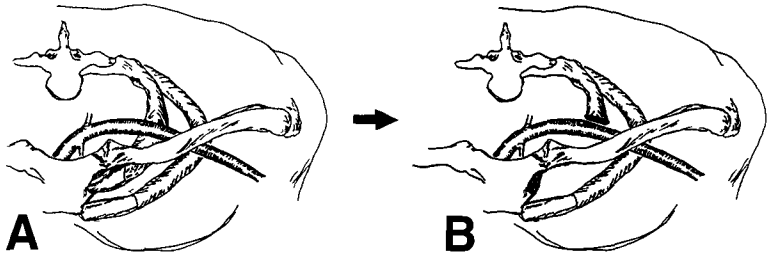

Fig 1. A, Top view of the thoracic outlet. **B,** Observe how, by removing the anterior portion of the first rib, the proximal portion of the axillary artery becomes an intrathoracic structure.